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Correlation of Electrocardiogram Derived P Wave Dispersion and Left Ventricular Diastolic Function in Hypertensive Patients

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العلاقة بين تشتت الموجة P من تخطيط كهربائية القلب والوظيفة الانبساطية للبطين الايسر عند مرضى ارتفاع ضغط الدم

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ABSTRACT

Background:

Abnormal PWD expressed the impaired atrial electrical conduction which occurs as a result of structural changes (remodelling) in the atria caused by hypertension (HT). Diastolic function of the left ventricle (LV) have significant role in determination LV filling. Diastolic dysfunction (DD) related with fatality and hospitalization due to heart failure (HF).

Patients and Methods:

The study involved 100 individuals with HT. PWD was calculated from 12- leads surface ECG by determining the disparity betwixt the broadest and thinnest P waves. LV mass and left ventricle mass index (LVMI) and diastolic function measurements were performed.

Results:

Correlation studies between PWD and parameters of LV diastolic function (E/A ratio and e`) showed statistically highly significant negative correlation ($r = - 0.401$, $r = - 0.244$) subsequently, $p < 0.05$).

Correlation study of PWD with E/e` showed a positive correlation which was statistically highly significant ($r = 0.241$, $p < 0.05$). While the Correlation studies of PWD with left atrial volume (LAV) and left atrial volume index (LAVI) showed statistically no significant positive correlation ($r = 0.116$, $r = 0.029$, $p > 0.05$).

Correlation studies of PWD with LVM and LVMI: showed statistically highly significant positive correlation with ($r = 0.474$, $r = 0.34$ respectively, $p < 0.05$).

Conclusions:

This research concludes that PWD obtained from ECG is a nonintrusive determiner for diastolic function in hypertensive patients, that can reflect LV diastolic function and the effects of hypertension on LV remodelling.

Keywords: P wave dispersion; impaired atrial electrical conduction; diastolic function; left ventricular remodelling; hypertension.

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INTRODUCTION

Arterial HT is hazardous cause for morbidness and fatality internationally, and is related with heightened risk of cardiovascular disease [1].

Blood pressure (BP) ≥ 130/80 mmHg in adults is considered HT, as higher BP is linked with the development of cardiovascular events, for instance stroke and myocardial infarction (MI) [2].

HT classified as primary (also called essential hypertension) and secondary HT. Primary HT forming 90% of HT in adults, with unknown cause and it is untreatable; Secondary HT forming 10% of HT in adults is induced by particular reasons for example: hyperparathyroidism, chronic kidney disease, hyperthyroidism, cushing syndrome, and several remedies, secondary HT is treatable [3].

HT is related with coronary heart disease and enhanced peril of stroke and associated with outcomes like HF, renal impairement, and retinal haemorrhage [3].

Risk factors for developing HT are modifiable risk factors and nonmodifiable risk factors. Modifiable risk factors involving smoking, alcohol consumption, diet, physical activity, BMI, abdominal obesity, stress, hyperglycemia and hypercholesterolemia, nonmodifiable risk factors such as age, gender, family history and ethnicity [4].

Diastole is a combination of physiological procedures that take place inside the heart throughout the filling of the ventricle and basically depend on the stiffness of the chamber and myocardial relaxation. Atypical diastolic function is linked with diverse cardiovascular disease mechanisms and is prognosticative for health issues [5].

DD and the related pathophysiological mechanisms that affect LV and LA structure and function is diagnosed by echocardiography [6].

PWD was measured on 12-leads ECG by determining P min wave period and P max wave period and then calculated by subtracting the P min from P max in any of the 12 leads [7]. PWD was increased in patients with LVDD [8] .

Hypertensive patients assessed for LVDD and its relation with PWD in non-dipper and dipper hypertensive patients. The conclusion was LVDD in dipper and non-dipper HT is associated with PWD [9].

PWD is enhanced in hypertensive patients with DD, and this enhancement is associated with the severeness of DD [10].

The aim of this research is to study the role of P wave dispersion (PWD) that is obtained from electrocardiogram (ECG) in detection of left ventricular diastolic dysfunction (LVDD) in hypertensive patients in relationship with diastolic parameters from echocardiography.

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PATIENTS AND METHODS

This cross sectional study involved 100 individuals diagnosed with HT (males and females), ranging in age from 20 to 40 years. This study was conducted in Merjan hospital in Al-Hilla City, during the time from September 2023 until June 2024. The study was done using echocardiography (PHILIPS Affinity 70C ultrasound machine). Doppler and 2D images were taken for all patients.

The biggest LA volume at the end of the systolic phase, where method of simpson was utilized for calculating LA volume, as seen in **[Fig. 1]**.

The E wave was detected by Pulsed wave Doppler at the mitral valve's leaflet tips, as shown in **[Fig. 2]**. And the ratio E/A was determined.The average of e' of the lateral and medial sides was measured by doppler imaging of tissue, where sample volume was placed at the mitral annulus from both the lateral and septal sides, as shown in **[Fig. 3]**, then the ratio E/e' was computed.

LV mass was measured by the following formula, LV mass (g) = 0.8 x {1.04 x [(IVST + LVIDD + PWT)^3 – (LVIDD)^3] } + 0.6 g, where IVST is the end-diastolic interventricular septal wall thickness, PWT is end-diastolic LV posterior wall thickness and LVIDD is LV internal end-diastolic dimension [11].

LVMI was calculated by dividing LVM by the body surface area (BSA), according to following equation: LVMI = LVM / BSA, where: LVMI represent left ventricular mass index, LVM represent left ventricular mass, BSA represent body surface area.

P wave dispersion calculated in milliseconds on the ECG with 12 leads, by determining P max wave period and P min wave period and then substracting the period of P min wave from the period of P max wave.

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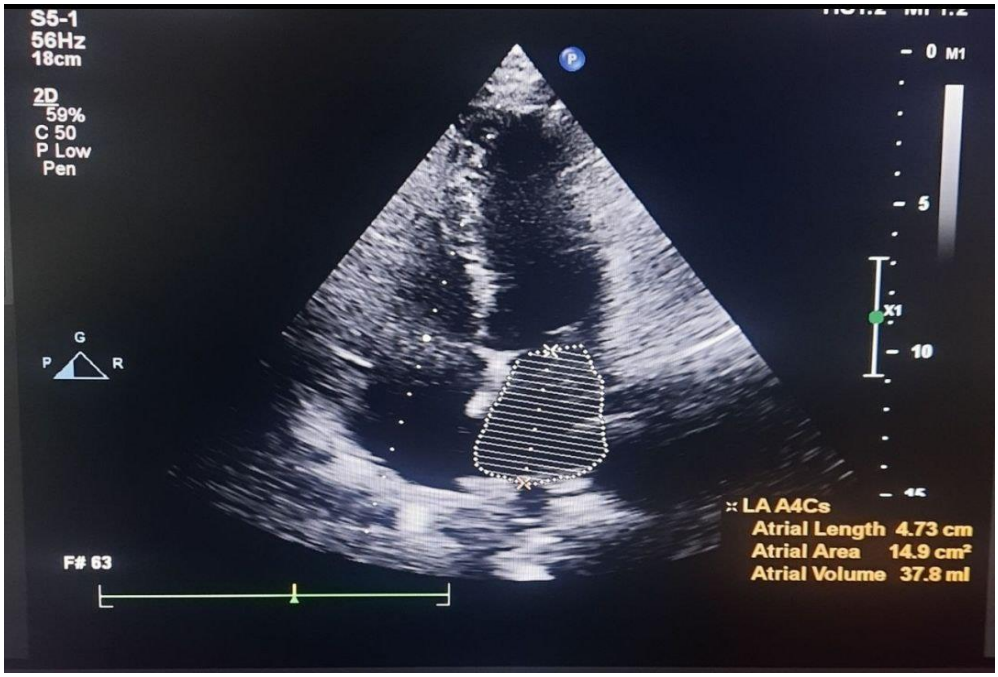

Figure (1): Left atrial volume measurement when the left atrial volume is the biggest, at the end of the systolic phase by simpson's method.

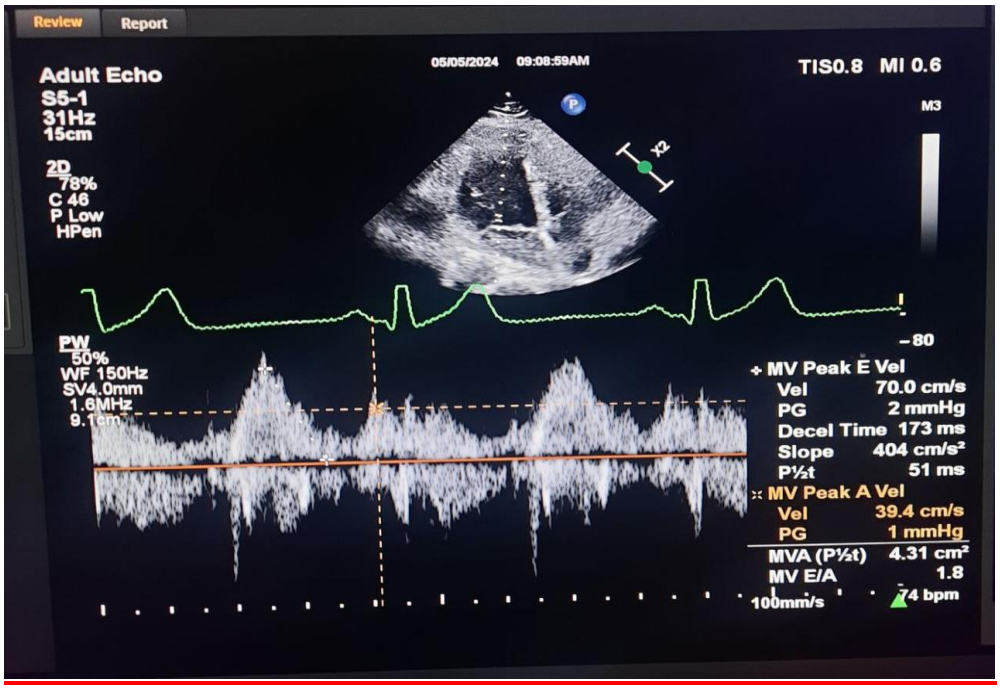

Figure (2): At the mitral valve's leaflet tips the pulsed wave doppler was utilized to detect peak velocity of early diastolic flow of the mitral valve.

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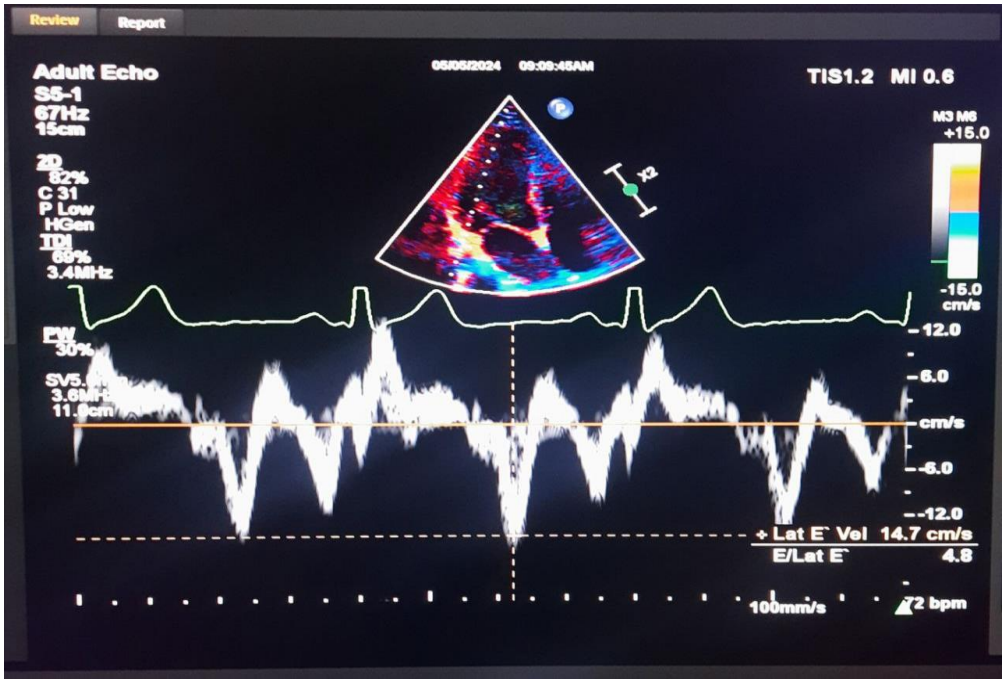

Figure (3): The mean of early diastolic peak velocity of mitral valve annulus of the lateral and septal sides was estimated by doppler imaging of tissue, where sample volume was located at the mitral annulus from both the lateral and septal sides, then the ratio of peak velocity of mitral valve early diastolic flow to early diastolic peak velocity of mitral valve annulus was calculated.

(E: peak velocity of early diastolic flow of the mitral valve, e`: early diastolic peak velocity of mitral valve annulus).

RESULTS AND DISCUSSION

All anthropometric data and echocardiographic are presented as mean ± SD in **[Table. 1]**.

Table (1): Anthropometric and 2D-echocardiographic data of hypertensive patients. Values are presented as mean ± SD.

Parameters	**Patient group N = 100 Mean ± SD**
Age (year)	43.95 ± 7.728
BMI(Kg/m^2)	32.593 ± 5.808
BSA(m^2)	2.007480 ± 0.24422
P wave dispersion(ms)	43.59 ± 5.196
E/A	1.008 ± 0.295
e` prime (cm/s)	10.51 ± 3.079
E/e` prime	7.382 ± 2.41
LA volume (ml)	40.158 ± 11.90
LA volume index (ml/m^2)	20.19 ± 6.240
TR velocity (m/s)	1.05 ± 1.517
LVM (g)	145.54 ± 35.515
LVMI (g/m^2)	72.278 ± 15.329

BMI: body mass index, BSA: body surface area, E/A: ratio of peak velocity of early diastolic flow of the mitral valve to peak velocity of late diastolic flow of the mitral valve, e`: early diastolic peak velocity of mitral valve annulus, LA volume: left atrial volume, LA volume index: left atrial volume index, TR velocity: tricuspid regurgitation velocity, LVM: left ventricular mass, LVMI: left ventricular mass index. SD: standard deviation, N: number of patients.

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Table (2): Correlations of P wave dispersion with 2D echocardiographical parameters.

Parameters	**PWD**
E/A Pearson correlation P value	- 0.401 0.000
e` Pearson correlation P value	- 0.244 0.001
E/e` Pearson correlation P value	0.241 0.001
LAV(ml) Pearson correlation P value	0.116 0.121
LAVI(ml/m^2) Pearson correlation P value	0.029 0.695
TR velocity Pearson correlation P value	0.015 0.84
LVM Pearson correlation P value	0.474 0.000
LVMI Pearson correlation P value	0.34 0.000

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E/A: ratio of peak velocity of early diastolic flow of the mitral valve to peak velocity of late diastolic flow of the mitral valve, e`: early diastolic peak velocity of mitral valve annulus, E/e`: ratio of peak velocity of early diastolic flow of the mitral valve to early diastolic peak velocity of mitral valve annulus, LAV: left atrial volume, LAVI: left atrial volume index, TR velocity: tricuspid regurgitation velocity, LVM: left ventricular mass, LVMI: left ventricular mass index.

Correlation study betwixt PWD and LV diastolic parameter E/A ratio showed negative correlation which was statistically significant (r = - 0.401, P value = 0.000), as shown in [Fig. 4].

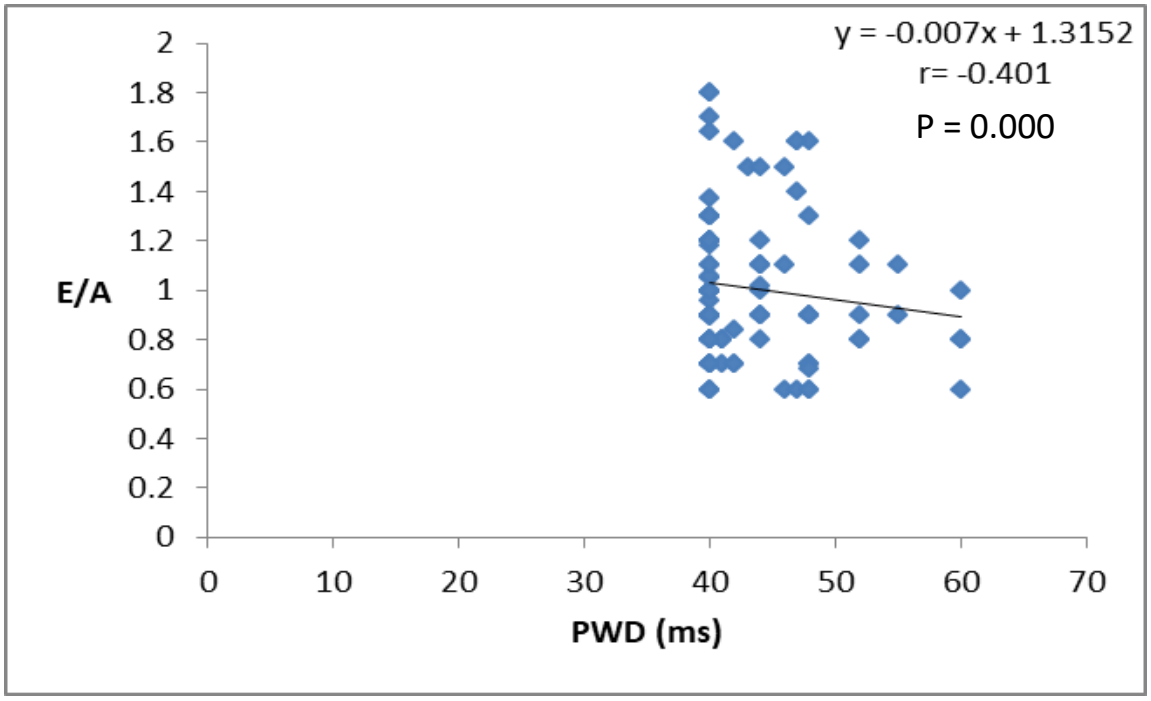

Figure (4): Correlation study of Electrocardiogram derived P wave dispersion and echocardiographic pulse Doppler derived mitral flow: peak velocity of early diastolic flow of the mitral valve to peak velocity of late diastolic flow of the mitral valve.

(PWD: P wave dispersion, E: peak velocity of early diastolic flow of the mitral valve A: peak velocity of late diastolic flow of the mitral valve).

Correlation study between PWD and LV diastolic parameter e` showed statistically significant negative correlation (r = - 0.244, P value = 0.001), as shown in [Fig. 5].

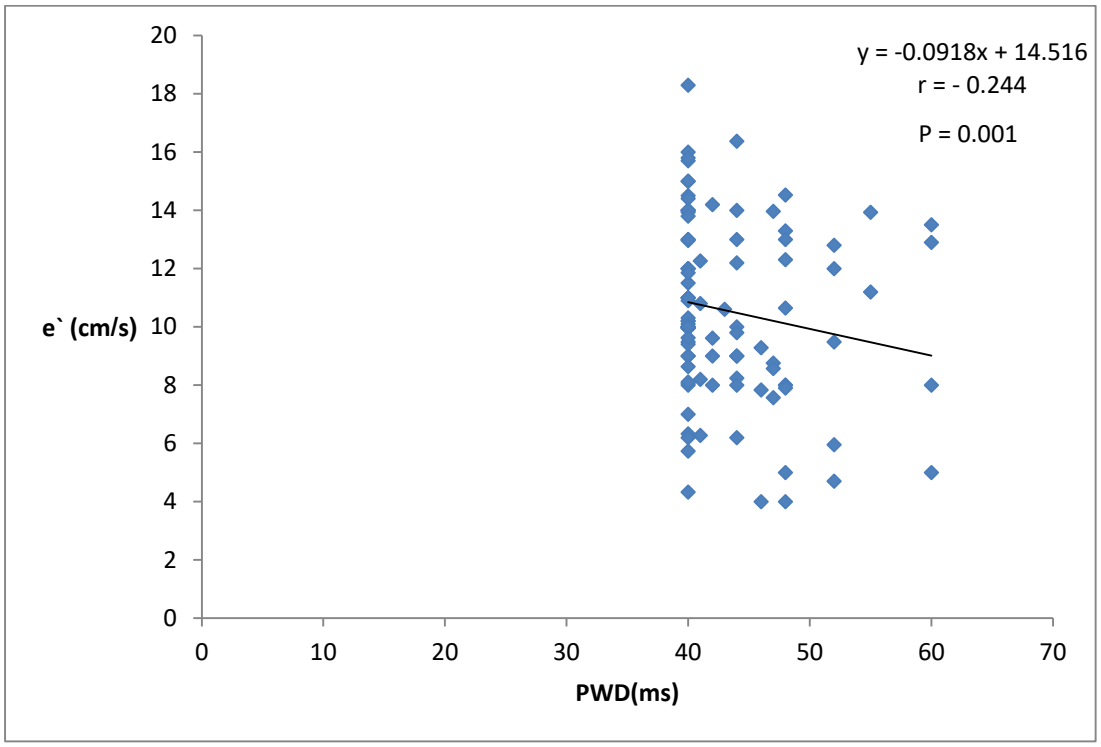

Figure (5): Correlation study of electrocardiogram derived P wave dispersion and echocardiographic tissue Doppler derived left ventricular early diastolic peak velocity of mitral valve annulus.

(PWD: P wave dispersion, e`: early diastolic peak velocity of mitral valve annulus).

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Correlation study of PWD with mitral valve E/e` showed a positive correlation which was statistically highly significant (r = 0.241, P = 0.001), as shown in [Fig. 6].

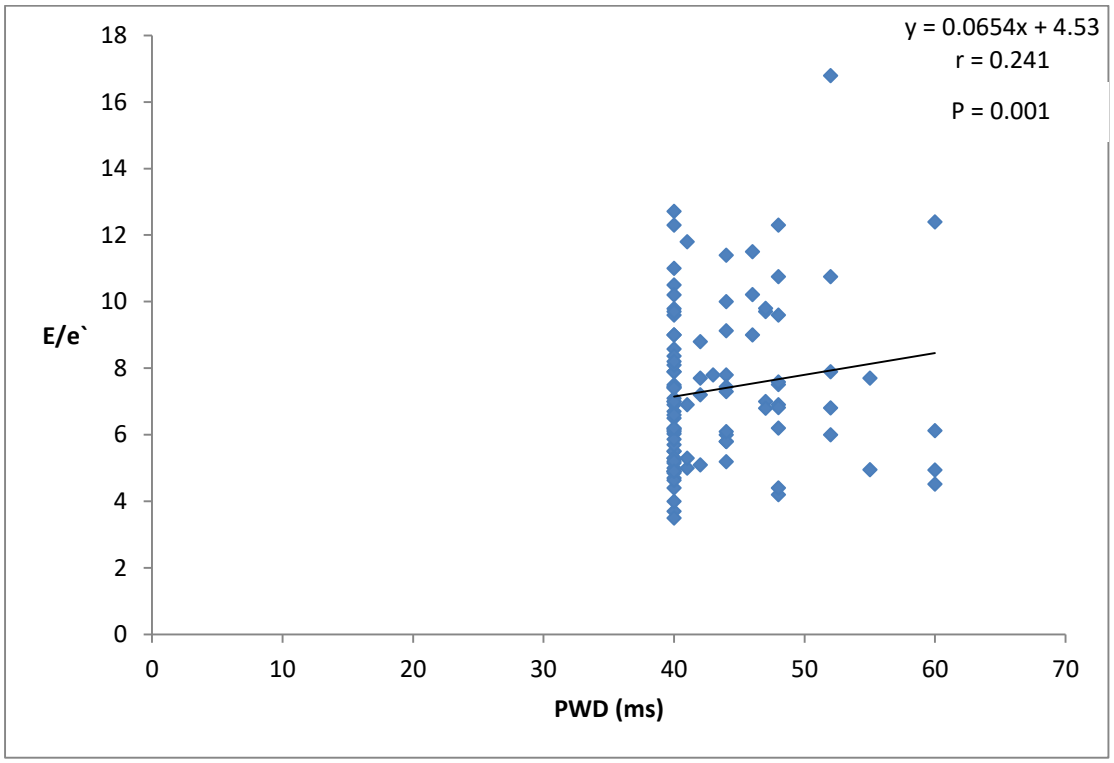

Figure (6): Correlation study of electrocardiogram derived P wave dispersion and echocardiographic derived peak velocity of early diastolic flow of the mitral valve to early diastolic peak velocity of mitral valve annulus.

(PWD: P wave dispersion, E: peak velocity of early diastolic flow of the mitral valve, e`: mitral valve annulus early diastolic peak velocity).

Correlation of PWD with LAV, LAVI and TR velocity showed statistically non-significant positive correlation (r = 0.116, 0.029 and 0.015 respectively, p > 0.05), [Table. 2].

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Correlation study of PWD with LVM: showed a positive correlation which was statistically significant (r = 0.474, P = 0.000), as shown in **[Fig. 7]**.

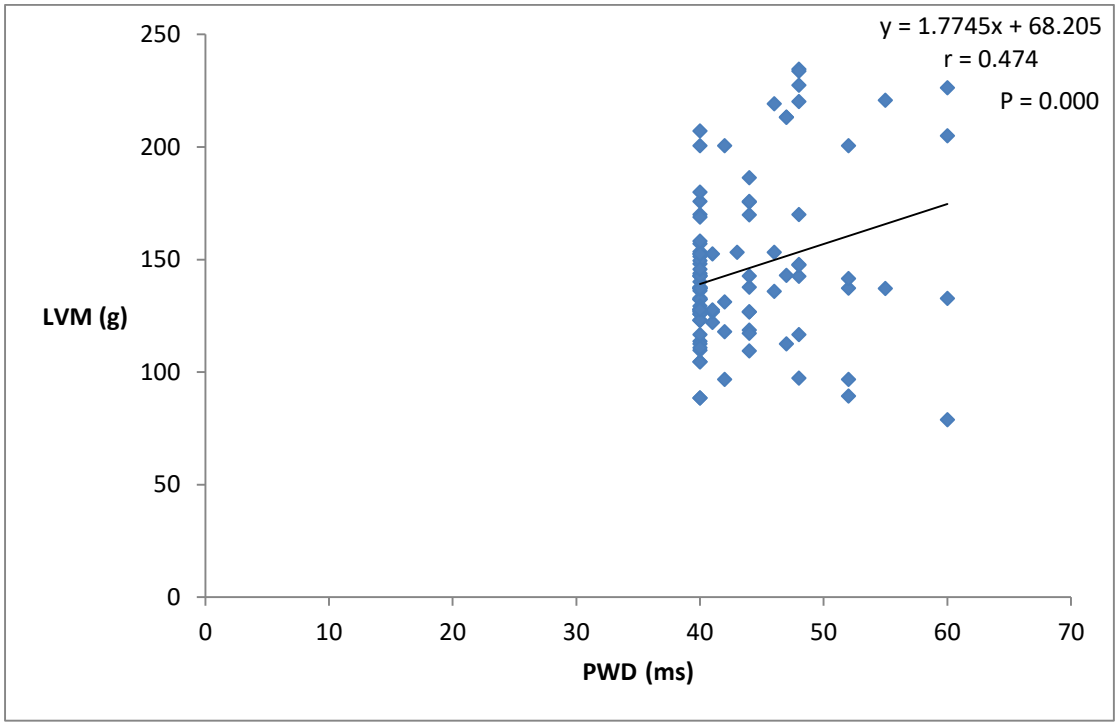

Figure (7): Correlation study of electrocardiogram derived P wave dispersion and echocardiographic tissue Doppler derived left ventricular mass.

(PWD: P wave dispersion, LVM: Left ventricular mass).

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Correlation study of PWD with LVMI: showed a positive correlation which was statistically significant (r = 0.34, P = 0.000), as shown in [Fig. 8].

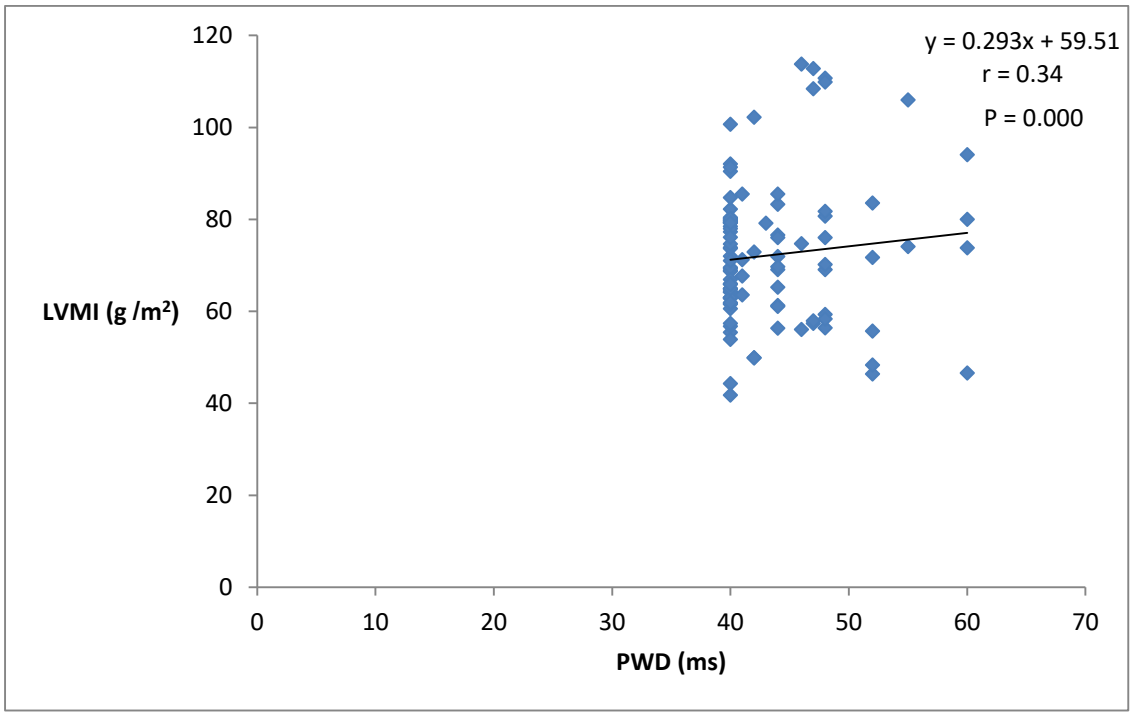

Figure (8): Correlation study of electrocardiogram derived P wave dispersion and echocardiographic tissue Doppler derived left ventricular mass index.

(PWD: P wave dispersion, LVMI: Left ventricular mass index).

This study showed that the correlation studies between PWD and LV diastolic parameters E/A ratio and e` was statistically significant negative correlation. This means PWD is inversely and significantly correlated with diastolic parameters in this study. This correlation may be resulted from the effects of hemodynamical and structural changes of the LVDD on left atrial structure and function in hypertensive patients [8]. This study is in agreement with another study that showed significant negative correlation between PWD and diastolic parameters [12]. Longer PWD duration correlated significantly with the parameters of impaired diastolic function [13]. Correlation studies of PWD with mitral valve E/e` showed statistically highly significant positive correlation, which means PWD is positively and significantly correlated with mitral valve E/e` in this study, where impaired relaxation of the LV in DD in which e` is decreased lead to increased mitral valve E/e`. PWD also increased positively and significantly with the increase of E/e` in DD due to elevated pressure inside LA that lead to structural changes ( remodelling ) in the LA which result in heterogeneous electrical conduction of the LA [14]. Another study showed that the E/e` is higher than normal in hypertensive patients with DD and having prolonged PWD in which the correlation between PWD and E/e` was positive nonsignificant correlation [8]. Correlation of PWD

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with LVM and LVMI in this study showed statistically significant positive correlation, which means PWD is positively and significantly correlated with LVM and LVMI. Increased LVM and LVMI in hypertensive patients duo to enlarged exertion on the ventricular wall which in turn lead to increased ventricular wall mass to equalize the stress on LV wall, the increased ventricular wall mass lead to increased ventricular wall stiffness and decreased the compliance leading to impaired relaxation which is the first stage of DD, Impaired relaxation of LV in DD lead to increased pressure inside the LA which causes structural changes [15] that affect LA electrical conduction that appear as prolonged PWD on ECG [14 .

CONCLUSIONS:

This study concludes that PWD obtained from ECG is a nonintrusive determinant of hypertensive patient's diastolic function,that can reflect LV diastolic function and hypertension effects on LV remodelling.

Conflict of interests.

There is no conflict of interests.

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الخلاصة

الهدف من الدراسة: دراسة دور تشتت الموجة P من تخطيط كهربائية القلب في الكشف عن اختلال الوظيفة الانبساطية للبطين الايسر عند مرضى ارتفاع ضغط الدم في علاقة مع المقاييس الانبساطية من تخطيط صدى القلب.

المقدمة: تشتت الموجة **P** الغير طبيعي يعبر عن الضعف في التوصيل الكهربائي في الأذين الايسر و الذي يحدث كنتيجة للتغيرات الهيكلية في الاذين بسبب ارتفاع ضغط الدم. الوظيفة الانبساطية للبطين الايسر لها دور مهم في تحديد ملئ البطين الايسر.اختلال الوظيفة الانبساطية له علاقة مع الوفاة و دخول المستشفى بسبب فشل القلب.

المرضى و طرق العمل: الدراسة شملت 100 شخص مصاب بأرتفاع ضغط الدم. تشتت الموجة P تم حسابه من تخطيط كهربائية القلب ذات 12 قطب, من خلال ايجاد الفرق بين اطول موجة P و اقصر موجة P . تم حساب قياسات كتلة البطين الايسر ومؤشر كتلة البطين الايسر و الوظيفة الانبساطية.

النتائج: دراسات العلاقة بين تشتت الموجة P و المقاييس الانبساطية للبطين الايسر E/A , e` اظهرت علاقة عكسية ذات قيمة كبيرة احصائيا ($r = -0.401$, $r = -0.244$ على التوالي $P < 0.05$).

دراسة علاقة تشتت الموجة P مع E/e` اظهرت علاقة طردية ذات قيمة كبيرة احصائيا ($r = 0.241$, $P < 0.05$).

بينما دراسات علاقة تشتت الموجة P مع حجم الأذين الايسر و مؤشر حجم الأذين الايسر, اظهر علاقة طردية ذات قيمة غير كبيرة احصائيا ($r = 0.116$, $r = 0.029$, $P > 0.05$).

اظهرت دراسات علاقة تشتت الموجة P مع كتلة البطين الايسر و مؤشر كتلة البطين الايسر علاقة طردية ذات قيمة كبيرة احصائيا ($r = 0.474$, $r = 0.34$ على التوالي, $P < 0.05$).

الاستنتاج: الدراسة استنتجت بأن تشتت الموجة P من تخطيط كهربائية القلب هي محدد غير تدخلي للوظيفة الانبساطية عند مرضى ارتفاع ضغط الدم و الذي يعكس الوظيفة الانبساطية للبطين الايسر و تأثيرات ارتفاع ضغط الدم على اعادة تشكيل البطين الايسر.

الكلمات المفتاحية: تشتت الموجة P ; ضعف التوصيل الكهربائي الاذيني ; الوظيفة الانبساطية ; التغير في بنية البطين الايسر ; ارتفاع ضغط الدم.

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